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2005 MeSH

MeSH Supplementary Concept Data

Return to Entry Page

Name of Substance	fluoresceinyl-arginyl-glutamyl-aspartyl-glutamyl-aspartyl-glutamyl-isoleucyl-glutamyl-tryptophan
Record Type	C
Registry Number	0
Entry Term	fluoresceinyl-REDEDEIEW
Entry Term	fluoresceinyl-Arg-Glu-Asp-Glu-Asp-Glu-Ile-Glu-Trp
Entry Term	Fl-CDB3
Heading Mapped to	*Fluoresceins
Heading Mapped to	*Oligopeptides
Source	Proc Natl Acad Sci U S A 2003 Nov 11;100(23):13303-7
Frequency	1
Note	binds and stabilizes the tumor suppressor p53 core domain, thereby preventing denaturation; may serve as an antineoplastic agent
Date of Entry	20031226
Unique ID	C479932

Return to Entry Page   **Link to NLM Cataloging Classification**

Refine Search

Search Results -

Term	Documents
@PY	8218647
(20 AND (@PY < "2001")).PGPB,USPT.	22
(L20 AND @PY<2001).PGPB,USPT.	22

Database:

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DB=PGPB,USPT; THES=ASSIGNEE; PLUR=YES; OP=ADJ

Set Name	Query	Hit Count	Set Name
L21	L20 and @py<2001	22	L21
L20	L19 and L5	200	L20
L19	binding protein same p53	851	L19
L18	L17 and p53	77	L18
L17	L16 and @py<2001	77	L17
L16	L15 and stabil$	77	L16
L15	L14 and denaturation	122	L15
L14	L13 and tumor	412	L14
L13	L12 and @py<2001	480	L13
L12	L5 and L8	3625	L12
L11	L10 and L9 and L8 and L5	0	L11
L10	prevent denaturation	430	L10

L9	antineoplastic agent	4423	L9
L8	p53	11124	L8
L7	L6 and tumor sepressor	0	L7
L6	L5 and p53	3625	L6
L5	Fluoresceins	33488	L5
L4	fluorescinyl-Arg-Glu-Asp-Glu-Asp-Glu-Ile-Glu-Trp	0	L4
L3	Fluoresceinyl-REDEDEIEW	0	L3
L2	REDEDEIEW	1	L2
L1	CDB3	7	L1

END OF SEARCH HISTORY

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                 alerts (SDIs) affected
NEWS 11 DEC 17 SOLIDSTATE reloaded; updating to resume; current-awareness
                 alerts (SDIs) affected
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                 alerts (SDIs) affected
NEWS 13 DEC 17 THREE NEW FIELDS ADDED TO IFIPAT/IFIUDB/IFICDB
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NEWS 15 DEC 30 CAPLUS - PATENT COVERAGE EXPANDED
NEWS 16 JAN 03 No connect-hour charges in EPFULL during January and
                 February 2005
NEWS 17 FEB 25 CA/CAPLUS - Russian Agency for Patents and Trademarks
                 (ROSPATENT) added to list of core patent offices covered
NEWS 18 FEB 10 STN Patent Forums to be held in March 2005
NEWS 19 FEB 16 STN User Update to be held in conjunction with the 229th ACS
                 National Meeting on March 13, 2005
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                 data from INPADOC
NEWS 21 FEB 28 BABS - Current-awareness alerts (SDIs) available
NEWS 22 FEB 28 MEDLINE/LMEDLINE reloaded
NEWS 23 MAR 02 GBFULL: New full-text patent database on STN
NEWS 24 MAR 03 REGISTRY/ZREGISTRY - Sequence annotations enhanced
NEWS 25 MAR 03 MEDLINE file segment of TOXCENTER reloaded

NEWS EXPRESS JANUARY 10 CURRENT WINDOWS VERSION IS V7.01a, CURRENT
              MACINTOSH VERSION IS V6.0c(ENG) AND V6.0Jc(JP),
              AND CURRENT DISCOVER FILE IS DATED 10 JANUARY 2005

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FILE COVERS 1907 - 8 Mar 2005 VOL 142 ISS 11
FILE LAST UPDATED: 7 Mar 2005   (20050307/ED)

   This file contains CAS Registry Numbers for easy and accurate
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=> s friedler a/au
L1   3 FRIEDLER A/AU

=> d L1 1-3 ibib,abs

L1   ANSWER 1 OF 3   CAPLUS   COPYRIGHT 2005 ACS on STN
ACCESSION NUMBER: 2002:101989 CAPLUS
DOCUMENT NUMBER: 136:303881
TITLE: Reduction in the incidence of type 2 diabetes with
                            lifestyle intervention or metformin
AUTHOR(S):   Knowler, William C.; Barrett-Connor, Elizabeth;
                            Fowler, Sarah E.; Hamman, Richard F.; Lachin, John M.;
                            Walker, Elizabeth A.; Nathan, David M.; Bray, G. A.;
                            Culbert, I. W.; Champagne, C. M.; Crow, M. D.; Dawson,
                            L.; Eberhardt, B.; Greenway, F. L.; Guillory, F. G.;
                            Herbert, A. A.; Jeffirs, M. L.; Joyce, K.; Kennedy, B.
                            M.; Lovejoy, J. C.; Mancuso, S.; Melancon, L. E.;
                            Morris, L. H.; Reed, L.; Perault, J.; Rau, K.; Ryan,
                            D. H.; Sanford, D. A.; Smith, K. G.; Smith, L. L.;
                            Smith, S. R.; St. Amant, J. A.; Terry, M.; Tucker, E.;
                            Tulley, R. T.; Vicknair, P. C.; Williamson, D.;
                            Zachwieja, J. J.; Ehrmann, D. A.; Matulik, M. J.;
                            Clark, B.; Collins, D. A.; Czech, K. B.; DeSandre, C.;
                            Geiger, G.; Frief, S.; Harding-Clay, B.; Hilbrich, R.
                            M.; Le Grange, D.; McCormick, M. R.; McNabb, W. L.;
                            Polonsky, K. S.; Sauter, N. P.; Semenske, A. R.;

Stepp, K. A.; Tobian, J. A.; Watson, P. G.; Mendoza,
J. T.; Smith, K. A.; Caro, J.; Goldstein, B.; Lark,
C.; Menefee, L.; Murphy, L.; Pepe, C.; Spandorfer, J.
M.; Goldberg, R. B.; Rowe, P.; Calles, J.; Casanova,
P.; Donahue, R. P.; Florez, H. J.; Giannella, A.;
Larreal, G.; McLymont, V.; Mendez, J.; O'Hara, P.;
Ojito, J.; Prineas, R.; Saab, P. G.; Haffner, S. M.;
Montez, M. G.; Lorenzo, C.; Miettinen, H.; Mobley, C.
M.; Mykkanen, L. A.; Rozek, M. M.; Hamman, R. F.;
Nash, P. V.; Testaverde, L.; Anderson, D. R.;
Ballonoff, L. B.; Bouffard, A.; Calonge, B. N.;
Farago, M.; Georgitis, W. J.; Hill, J. O.; Hoyer, S.
R.; Jortberg, B. T.; Merenich, J. A.; Miller, M.;
Regensteiner, J. G.; Seagle, H. M.; Smith, C. M.;
Steinke, S. C.; Van Dorsten, B.; Horton, E. S.;
Lawton, K. E.; Arky, R. A.; Bryant, M.; Burke, J. P.;
Caballero, E.; Callaghan, K. M.; Devlin, D.; Franklin,
T.; Ganda, O. P.; **Goebel-Fabbri, A. E.;**
Harris, M.; Jackson, S. D.; Jacobsen, A. M.; Kula, L.
M.; Kocal, M.; Ledbury, S.; Malloy, M. A.; Mullooly,
C.; Nicosia, M.; Oldmixon, C. F.; Pan, J.; Pomposelli,
C.; Quitongan, M.; Rubtchinsky, S.; Schweizer, D.;
Seely, E. W.; Simonson, D.; Smith, F.; Solomon, C. G.;
Tyson, J.; Warram, J.; Kahn, S. E.; Montgomery, B. K.;
Alger, M.; Allen, E.; Barrett, T.; Bhanji, D.; Cowan,
J.; Cullen, J.; Fujimoto, W. Y.; Katz, B.; Knopp, R.
H.; Lipkin, E. W.; Marr, M.; McCann, B. S.; Palmer, J.
P.; Schwartz, R. S.; Uyema, D.; Kitabachi, A. E.;
Murphy, M. E.; Applegate, W. B.; Bryer-Ash, M.; Coble,
J. H.; Crisler, A.; Cunningham, G.; Franklin, A. W.;
Frieson, S. L.; Green, D. L.; Imseis, R.; Kennedy, C.
L.; Lambeth, H. C.; Latif, K. A.; Lichtermann, L. C.;
McIntyre, M. D.; Nault, J. D.; Oktaei, H.; O'Toole, M.
L.; Ricks, H.; Rutledge, L. M. K.; Schussler, S. C.;
Sherman, A. R.; Smith, C. M.; Soberman, J. E.;
Stewart, K. J.; Van Brunt, D. L.; Williams-Cleaves, B.
J.; Johnson, M. K.; Behrends, C.; Cook, M. L.;
Fitzgibbon, M.; Giles, M. M.; Heard, D.; Johnson, C.;
Larsen, D.; Lowe, A.; Lyman, M.; McPherson, D.;
Molitch, M. E.; Pitts, T.; Reinhart, R.; Roston, S.;
Schinleber, P. A.; Nathan, D. M.; McKitrick, C.;
Abbott, K.; Anderson, E.; Bissett, L.; Cagliero, E.;
Crowell, S.; Delahanty, L.; Fritz, S.; Hayward, K.;
Levina, E.; Michel, T.; Norman, D.; O'Keefe, J.;
Poulos, A.; Ronan, L.; Rosal, M.; Salerno, M.;
Schneider, M.; Shagensky, C.; Steiner, B.; Turgeon,
H.; Young, A.; Olefsky, J. M.; Carrion-Petersen, M.
L.; Barrett-Connor, E.; Beltran, M.; Caenepeel-Mills,
K.; Edelman, S. V.; Ford, R. O.; Garcia, J.; Hagerty,
M.; Henry, R. R.; Hill, M.; Horne, J.; Leos, D.;
Matney, J.; Mudaliar, S.; Petersen, G.; Pollard, A.;
Polonsky, W.; Szerdi, S.; Torio-Hurley, J.; Vejvoda,
K.; Pi-Sunyer, F. X.; Lee, J. E.; Allison, D. B.;
Agharanya, N.; Aronoff, N. J.; Baldo, M.; Foo, S. T.;
Hagamen, S.; Pal, C.; Parkes, K.; Pena, M.; Van Wye,
G. E. H.; Marrero, D. G.; Kukman-Kelly, M. S.; Dorson,
Y. F.; Fineberg, S. E.; Guare, J. C.; Hadden, A.;
Hills, B.; Ignaut, J. M.; Jackson, M. A.; Kirkman, M.
S.; Mather, K.; McAree, G.; Porter, B. D.; Prince, M.
J.; Wheeler, M. L.; Ratner, R. E.; Youssef, G.;
Shapiro, S.; Bonar, A.; Bronsord, M.; Brown, E.;
Cheatham, W. W.; Cola, S.; Comfort, A.; Boggs, G.;
Eagle, C.; Evans, C.; Gorman, E.; Johnson, R.;
Levetan, C.; Kellum, T.; Lagarda, M.; Nair, A. K.;

Passaro, M. D.; Phillips, W.; Saad, M. F.; Budgett,
M.; Fahmi, S.; Jinagouda, S. D.; Bernaba, B.; Bodkin,
S. L.; Ciobanu, V.; Commisso, R.; Cosenza, C.; Dinh,
T.; Gonzalez, M.; Khan, A.; Kumar, D.; Lui, G.; Mehra,
V.; Sharma, A.; Soukiazian, S.; Szamos, K.; Tramanian,
A.; Vargas, A.; Zambrana, N.; White, N. H.; Santiago,
A. S.; Das, S.; Brown, A. L.; Dagogo-Jack, S.; Fisher,
E. B.; Hurt, E.; Jones, T.; Kerr, M.; Ryder, L.;
Santiago, J. V.; Wernimont, C.; Saudek, C. D.;
Bradley, V. L.; Fowlkes, T.; Joseph, H.; Brancati, F.
L.; Charleston, J. B.; Clark, J. M.; Horak, K.;
Jiggetts, D.; Mosley, H.; Rubin, R. R.; Samuels, A.;
Stewart, K. J.; Thomas, L.; Williamson, P.; Schade, D.
S.; Adams, K. S.; Atler, L. F.; Bland, A.; Bowling, D.
A.; Boyle, P. J.; Burge, M. R.; Butler, L.; Canady, J.
L.; Chai, L.; Colleran, K. M.; Guillen, M.; Gonzales,
Y.; Gutierrez, M.; Hornbeck, D.; Johannes, C.; Karz,
P.; King, C.; Libby, E. N., III; McCalman, R.;
Montoya, D. A.; Rassam, A.; Rubinchik, S.; Senter, W.;
Shamoon, H.; Brown, J. O.; Adames, J.; Blanco, E.;
Cox, L.; Crandall, J. P.; Duffy, H.; Engel, S.;
Friedler, A.; Harroun, T.; Howard-Century, C. J.;
Kloiber, S.; Longchamp, N.; Pompi, D.; Violino, E.;
Walker, E. A.; Wylie-Rosett, J.; Zimmerman, E.;
Zonszein, J.; Wing, R. R.; Kramer, M. K.; Barr, S.;
Boraz, M. A.; Clifford, L.; Culyba, R.; Frazier, M.;
Gilligan, R.; Harris, L.; Harrier, S.; Henderson, W.;
Jeffreis, S.; Koenning, G.; Kriska, A. M.; Maholic,
K.; Manjoo, Q.; Mullen, M.; Noel, A.; Orchard, T. J.;
Orro, A.; Semler, L. N.; Smith, C.; Smith, M.;
Stapinski, V.; Viteri, J.; Wilson, T.; Williams, K.
V.; Zgibor, J.; Arakaki, R. F.; Latimer, R. W.;
Baker-Ladao, N. K.; Beddow, R. M.; Braginsky, R.;
Calizar, M.; Dias, L. M.; Durham, N.; Dupont, D. A.;
Fukuhara, L. L.; Inouye, J.; Mau, M. K.; Mikami, K.;
Mohideen, P.; Odom, S. K.; Sinkuie-Kam, B.; Tokunaga,
J. S.; Twiggs, R. U.; Wang, C. Y.; Vita, J.; Knowler,
W. C.; Cooeyate, N. J.; Hoskin, M. A.; Percy, C. A.;
Acton, K. J.; Andre, V. L.; Antone, S.; Baptisto, N.
M.; Barber, R.; Segay, S.; Bennett, P. H.; Benson, M.
B.; Beyale, S.; Bird, E. C.; Broussard, B. A.; Chavez,
M.; Daeawyma, T. S.; Doughty, M. S.; Duncan, R.;
Edgerton, C.; Ghahate, J. M.; Glass, M.; Gohdes, D.;
Grant, W.; Hanson, R. L.; Horse, E.; Hughte, G.;
Ingraham, L. E.; Jackson, M. C.; Jay, P. A.; Kaskalla,
R. S.; Kessler, D.; Kobus, K. M.; Krakoff, J.; Manus,
C.; Morgan, T.; Nashboo, Y.; Nelson, J.; Pauk, G. L.;
Poirier, S.; Polczynski, E.; Reidy, M.; Roumain, J.;
Rowse, D. H.; Roy, R. J.; Sangster, S.; Sewemaenewa,
J.; Tonemah, D.; Wilson, C.; Yazzie, M.; Fowler, S.;
Brenneman, T.; Abebe, S.; Bain, R.; Bamdad, J.;
Callaghan, J.; Edelstein, S. L.; Gao, Y.; Grimes, K.
L.; Grover, N.; Hirst, K.; Jones, S.; Jones, T. L.;
Katz, R. J.; Lachin, J. M.; Orlosky, R.; Stimpson, C.
E.; Suiter, C.; Temprosa, M. G.; Walker-Murray, F. E.
M.; Garfield, S.; Eastman, R.; Fradkin, J.; Andres,
R.; Engelgau, M. M.; Venkat Narayan, K. M.;
Williamson, D. F.; Herman, W. H.; Marcovina, S. M.;
Aldrich, A.; Chandler, W. L.; Rautaharju, P. M.;
Pemberton, N. T.; Prineas, R.; Rautaharju, F. S. R.;
Zhang, Z.; Mayer-Davis, E. J.; Costacou, T.; Martin,
M.; Sparks, K. L.; O'Leary, D. H.; Funk, L. R. C.;
O'Leary, K. A.; Polak, J. F.; Stamm, E. R.;
Scherzinger, A. L.; Wing, R. R.; Gillis, B. P.;

Huffmyer, C.; Kriska, A. M.; Venditti, E. M.; Walker, E. A.; Harroun, T.; Ganiats, T. G.; Groessl, E. J.; Beerman, P. R.; David, K. M.; Kaplan, R. M.; Sieber, W. J.; Genuth, S. M.; Cahill, G. F.; Ferris, F. L., III; Gavin, J. R., III; Halter, J. B.; Wittes, J.; Henry, R. R.; Haffner, S. M.; Rubin, R. R.; Montgomery, B. K.; Ratner, R. E.; Herman, W. H.; Kahn, S. E.; Santiago, J. V.; Olefsky, J.; Wing, R. R.; Saudek, C.; Montez, M.; Kramer, K.; Hamman, R. F.; Knowler, W. C.; Goldberg, R. B.; Fujimoto, W. Y.; Charleston, J.; Nathan, D. M.

CORPORATE SOURCE:	Diabetes Prevention Program Coordinating Center, Washington Univ., Rockville, MD, 20852, USA
SOURCE:	New England Journal of Medicine (2002), 346(6), 393-403
	CODEN: NEJMAG; ISSN: 0028-4793
PUBLISHER:	Massachusetts Medical Society
DOCUMENT TYPE:	Journal
LANGUAGE:	English

AB Type 2 diabetes affects approx. 8 % of adults in the United States. Some risk factors - elevated plasma glucose concns. in the fasting state and after an oral glucose load, over-weight, and a sedentary lifestyle - are potentially reversible. We hypothesized that modifying these factors with a lifestyle-intervention program or the administration of metformin would prevent or delay the development of diabetes. We randomly assigned 3234 nondiabetic persons with elevated fasting and post-load plasma glucose concns. to placebo, metformin (850 mg twice daily), or a lifestyle-modification program with the goals of at least a 7 % weight loss and at least 150 min of phys. activity per wk. The mean age of the participants was 51 yr, and the mean body-mass index (the weight in kilograms divided by the square of the height in meters) was 34.0; 68 % were women, and 45 % were members of minority groups. The average follow-up was 2.8 yr. The incidence of diabetes was 11.0, 7.8, and 4.8 cases per 100 person-years in the placebo, metformin, and life-style groups, resp. The lifestyle intervention reduced the incidence by 58 % (95 % confidence interval, 48 to 66 %) and metformin by 31 % (95 % confidence interval, 17 to 43 %), as compared with placebo; the lifestyle intervention was significantly more effective than metformin. To prevent one case of diabetes during a period of three years, 6.9 persons would have to participate in the lifestyle-intervention program, and 13.9 would have to receive metformin. Lifestyle changes and treatment with metformin both reduced the incidence of diabetes in persons at high risk. The lifestyle intervention was more effective than metformin.

REFERENCE COUNT:	29 THERE ARE 29 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L1   ANSWER 2 OF 3   CAPLUS   COPYRIGHT 2005 ACS on STN

ACCESSION NUMBER:	1999:578782 CAPLUS
DOCUMENT NUMBER:	132:11558
TITLE:	Human immunodeficiency virus type 1 Vif-derived peptides inhibit the viral protease and arrest virus production
AUTHOR(S):	Gilon, C.; **Friedler, A.**; Baraz, L.; Blumenzweig, I.; Nussinuv, O.; Steinitz, M.; Kotler, M.
CORPORATE SOURCE:	Department of Organic Chemistry, The Hebrew University, Jerusalem, 91904, Israel
SOURCE:	Peptide Science: Present and Future, Proceedings of the International Peptide Symposium, 1st, Kyoto, Nov. 30-Dec. 5, 1997 (1999), Meeting Date 1997, 439-441. Editor(s): Shimonishi, Yasutsugu. Kluwer: Dordrecht, Neth.
	CODEN: 68BYA5
DOCUMENT TYPE:	Conference

LANGUAGE: English
AB   To study the effects of Vif derived peptides on HIV maturation and the
     autoprocessing of the Gag and Gag-Pol polyproteins, N terminal Vif
     polypeptides were synthesized and inhibition of viral proteases was
     assayed by ELISA. The results show that 4 out of the 11 peptides
     significantly inhibited viral proteases. Vif derived polypeptides also
     inhibited the autoprocessing of the Gag and Gag-Pol polyproteins in
     bacteria and eukaryotic cells. These data suggest that the Vif derived
     peptides form an attractive potential therapeutic agent for inhibition of
     HIV proteases during HIV-1 infection in humans.
REFERENCE COUNT: 4   THERE ARE 4 CITED REFERENCES AVAILABLE FOR THIS
                                    RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L1   ANSWER 3 OF 3   CAPLUS   COPYRIGHT 2005 ACS on STN
ACCESSION NUMBER: 1999:578657   CAPLUS
DOCUMENT NUMBER: 132:102441
TITLE: BCvir: backbone cyclic peptide, which mimics the
                             nuclear localization signal of human immunodeficiency
                             virus type 1 matrix protein, inhibits nuclear import
                             and virus production in non-dividing cells
AUTHOR(S):   Friedler, A.; Zakai, N.; Karni, O.; Broder,
                             Y. C.; Baraz, L.; Kotler, M.; Loyter, A.; Gilon, C.
CORPORATE SOURCE: Department of Organic Chemistry, Institute of
                             Chemistry, The Hebrew University of Jerusalem,
                             Jerusalem, 91904, Israel
SOURCE: Peptide Science: Present and Future, Proceedings of
                             the International Peptide Symposium, 1st, Kyoto, Nov.
                             30-Dec. 5, 1997 (1999), Meeting Date 1997, 70-72.
                             Editor(s): Shimonishi, Yasutsugu. Kluwer: Dordrecht,
                             Neth.
                             CODEN: 68BYA5
DOCUMENT TYPE: Conference
LANGUAGE: English
AB   A backbone cyclic NLS-mimetic peptide was found which inhibits nuclear
     import in invitro assay systems as well as HIV-1 replication in infected
     cultured cells. This peptide, BCvir, is resistant to proteolysis. BCvir
     and similar peptides are potential candidates for the development of a
     novel class of anti-viral drugs based on blocking nuclear import of viral
     genomes.
REFERENCE COUNT: 10   THERE ARE 10 CITED REFERENCES AVAILABLE FOR THIS
                                    RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

=> s Friedler Assaf
            5 FRIEDLER
           22 ASSAF
L2   0 FRIEDLER ASSAF
               (FRIEDLER(W)ASSAF)

=> s fluoresceinyl and pepetide and p53
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           14 PEPETIDE
            6 PEPETIDES
           20 PEPETIDE
               (PEPETIDE OR PEPETIDES)
        30076 P53
L3   0 FLUORESCEINYL AND PEPETIDE AND P53

=> s fluoresceins
L4   227 FLUORESCEINS

=> s binding peptide
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         1890 BINDINGS

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          416868 PEPTIDE
                 (PEPTIDE OR PEPTIDES)
L5          3659 BINDING PEPTIDE
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=> s p53 and binding protein
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         1192773 PROTEINS
         1996128 PROTEIN
                 (PROTEIN OR PROTEINS)
          134380 BINDING PROTEIN
                 (BINDING(W)PROTEIN)
L6          1376 P53 AND BINDING PROTEIN

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L2            0 S FRIEDLER ASSAF
L3            0 S FLUORESCEINYL AND PEPETIDE AND P53
L4          227 S FLUORESCEINS
L5         3659 S BINDING PEPTIDE
L6         1376 S P53 AND BINDING PROTEIN

=> s L6 and L5 and L4
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=> s L4 and L6
L8            0 L4 AND L6

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           93176 PREVENTS
          306930 PREVENT
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           34731 DENATURATION
                 (DENATURATION OR DENATURATIONS)
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                 (PREVENT(W)DENATURATION)
L9            0 L6 AND PREVENT DENATURATION

=> L6 and stabilizing molecule
L6 IS NOT A RECOGNIZED COMMAND
The previous command name entered was not recognized by the system.
For a list of commands available to you in the current file, enter
"HELP COMMANDS" at an arrow prompt (=>).

=> s L6 and stabilising molecule
              80 STABILISING
           46847 MOLECULE
          145652 MOLECULES
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         2520191 MOL
                    (MOL OR MOLS)
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                    (MOLECULE OR MOL)
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                    (STABILISING(W)MOLECULE)
L10            0 L6 AND STABILISING MOLECULE

=> d his

       (FILE 'HOME' ENTERED AT 08:50:59 ON 08 MAR 2005)

       FILE 'CAPLUS' ENTERED AT 08:51:08 ON 08 MAR 2005
L1             3 S FRIEDLER A/AU
L2             0 S FRIEDLER ASSAF
L3             0 S FLUORESCEINYL AND PEPETIDE AND P53
L4           227 S FLUORESCEINS
L5          3659 S BINDING PEPTIDE
L6          1376 S P53 AND BINDING PROTEIN
L7             0 S L6 AND L5 AND L4
L8             0 S L4 AND L6
L9             0 S L6 AND PREVENT DENATURATION
L10            0 S L6 AND STABILISING MOLECULE

=> s p53 and stabiliz molecule
MOLECULE IS NOT A RECOGNIZED COMMAND
The previous command name entered was not recognized by the system.
For a list of commands available to you in the current file, enter
"HELP COMMANDS" at an arrow prompt (=>).

=> s p53 and stabil? molecule
           30076 P53
          962138 STABIL?
           46847 MOLECULE
          145652 MOLECULES
          186045 MOLECULE
                    (MOLECULE OR MOLECULES)
         2198618 MOL
          590871 MOLS
         2520191 MOL
                    (MOL OR MOLS)
         2559007 MOLECULE
                    (MOLECULE OR MOL)
             640 STABIL? MOLECULE
                    (STABIL?(W)MOLECULE)
L11            2 P53 AND STABIL? MOLECULE

=> d L11 1-2 ibib,abs

L11  ANSWER 1 OF 2  CAPLUS  COPYRIGHT 2005 ACS on STN
ACCESSION NUMBER:        2003:133296  CAPLUS
DOCUMENT NUMBER:         138:166255
TITLE:                   Stabilization of the native conformation of a mutant
                         tumor suppressor protein p53 and other
                         mutant proteins using CDB3 peptide and other
                         biomolecules and application to treatment of cancer
                         and other diseases
INVENTOR(S):             Friedler, Assaf; Fersht, Alan
PATENT ASSIGNEE(S):      Medical Research Council, UK
SOURCE:                  PCT Int. Appl., 73 pp.
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PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2003014144	A2	20030220	WO 2002-GB3668	20020809
WO 2003014144	A3	20031127		

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN,
CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH,
GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR,
LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH,
PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TN, TR, TT, TZ,
UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW

RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY,
KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES,
FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, SK, TR, BF, BJ, CF,
CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG

| EP 1414846 | A2 | 20040506 | EP 2002-749128 | 20020809 |

R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT,
IE, SI, LT, LV, FI, RO, MK, CY, AL, TR, BG, CZ, EE, SK

| US 2005008653 | A1 | 20050113 | US 2004-775679 | 20040210 |

PRIORITY APPLN. INFO.:

	GB 2001-19557	A	20010810
	GB 2001-27917	A	20011121
	GB 2002-10740	A	20020510
	WO 2002-GB3668	W	20020809

AB   We disclose a method of stabilizing the native state of a polypeptide, the
     method comprising exposing the polypeptide to a **stabilizing
     mol.** capable of binding to the polypeptide at a site which at
     least partially overlaps a functional site in its native state.   The
     authors describe the isolation and identification of a stabilizing peptide
     CDB3, which is capable of binding the tumor suppressor protein **p53**
     near its DNA binding site, and stabilizing the native form of the protein.
     Since the binding of DNA itself stabilizes **p53** core domain, and
     it binds very tightly, stabilization by a peptide such as CDB3 is needed
     only for mutants where DNA binding is impaired because mutant **p53**
     is in denatured conformation.   Once the protein has bound DNA, the peptide
     is not needed any more.   The ability of CDB3 to induce refolding of
     p53 core domain, together with the observation that DNA can
     displace it from **p53**, led the authors to propose the a
     "chaperone" mechanism for rescuing a denatured oncogenic protein: CDB3
     binds only the native state of the oncogenic protein which is able to bind
     DNA, probably immediately on biosynthesis, and therefore shifts the equilibrium
     towards the native state.   Then DNA can bind the protein, displacing the
     peptide, which is free again to bind another protein mol.   Exemplary
     design of potential **P53** core domain binding peptides, screening
     of the CDB peptides for binding **p53** core domain, identification
     of the **P53** core domain binding peptide CDB3, characterization of
     CDB3-**P53** core domain binding and binding of fluorescein-labeled
     CDB3 are reported.   **Stabilizing mols.** and/or compns.
     of the invention can be used in the treatment of any animal or human
     disease where errors in protein conformation, folding and aggregation
     contribute to the disease.   Examples include cancer, cystic fibrosis and
     neuro-degeneration.   In a particularly preferred embodiment, the disease
     is cancer.

Fuchs, Serge Y.; Adler, Victor; Mai, Masyoshi; Ronai, Ze'ev

AB   Identification of Mdm2 and JNK as proteins that target degradation of
      wild-type (wt) p53 prompted the examination of their effect on mutant
      p53, which exhibits a prolonged half-life. Of five mutant
      p53 forms studied for association with the targeting mols., two no
      longer bound to Mdm2 and JNK. Three mutant forms, which exhibit high
      expression levels, showed lower affinity for association with Mdm2 and JNK in
      concordance with greater affinity to p14ARF, which is among the
      stabilizing p53 mols. Monitoring mutant p53 stability
      in vitro confirmed that, whereas certain forms of mutant p53 are
      no longer affected by either JNK or Mdm2, others are targeted for degradation
      by JNK/Mdm2, albeit at lower efficiency when compared with wt p53
      . Expression of wt p53 in tumor cells revealed a short
      half-life, suggesting that the targeting mols. are functional. Forced
      expression of mutant p53 in p53 null cells confirmed
      pattern of association with JNK/Mdm2 and prolonged half-life, as found in the
      tumor cells. Over-expression of Mdm2 in either tumor (which do express
      endogenous functional Mdm2) or in p53 null cells decreased the
      stability of mutant p53 suggesting that, despite its expression,
      Mdm2/JNK are insufficient (amount/affinity) for targeting mutant p53
      degradation Based on both in vitro and in vivo analyses, the prolonged
      half-life of mutant p53 depends on the nature of the mutation,
      which either alters association with targeting mols., ratio between
      p53 and targeting/stabilizing mols., or
      targeting efficiency. (c) 2000 Academic Press.